# TREATING PSYCHOLOGICAL TRAUMA AND PTSD

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# *Treatment Goals for PTSD*

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It has been more than 20 years since the term "posttraumatic stress disorder" (PTSD) was included in the third edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-III) of the American Psychiatric Association (1980). The inclusion of PTSD under the rubric of the anxiety disorders was not without political controversy, academic and theoretical debate, or outright skepticism as to its scientific validity as an illness (see Krystal, 1968, and Wilson, 1994, for reviews). Despite the catastrophic stress-related events which served to define nodal world crisis points in the 20th century (e.g., World War I; World War II; the Holocaust; Hiroshima and Nagasaki; state terrorism and political tyranny; the Korean War; the Vietnam War; the Cambodian genocide and more recent ethnic massacres in Bosnia, Rwanda, Kosovo, and East Timur; technological disasters such as that in Bhopal, India; and the threat of nuclear accidents such as the meltdown and explosion at Chernobyl), the *absence* of a separate diagnostic category for trauma-related psychiatric syndromes was simply a fact from DSM-I (American Psychiatric Association, 1952) to DSM-III (American Psychiatric Association, 1980). Beyond a doubt these historical and tragic catastrophic events had life-altering sequelae to persons, cultures, governments, and nations. It is only reasonable, therefore, that the absence of a specific diagnostic category for PTSD had medical and psychiatric consequences for the quality of health care and treatment of trauma victims. While it is possible to speculate on the reasons for this void in scientific and medical classification, the advent of PTSD as a separate diagnostic category (American Psychiatric Association, 1980) was a distinct and critical turning point in the advancement of knowledge. Today the legacy of these traumatic experiences are still present in

memory, spirit, and being for many individuals who persist in their search for meaning in an effort to understand their victimization within the fabric of modern civilization.

In many respects it remains a puzzle that a "cloistered" group of mental health professionals charged with the responsibility of revising the psychiatric criteria of DSM-I (American Psychiatric Association, 1952) (i.e., DSM-II 1968 APA publication committee) would have difficulty in recognizing and accepting the necessity of scientifically classifying PTSD as a distinct psychiatric disorder, especially given the profound worldwide and historic traumatic events which punctuated the 20th century. Moreover, prior to DSM-II, there was voluminous scientific literature on traumatic stress (see Laughlin, 1967, for a review). We have to wonder, collectively and individually, why it took so long to acknowledge "officially" that psychic trauma can lead to a distinct psychiatric illness of a chronic nature or cause alterations in personality functioning which may be pathological or transformative in ego functioning and identity (Wilson, 1988). Indeed the field of stress medicine and psychoanalytic investigations established parameters of prolonged stress effects to the organism well before PTSD was classified in DSM-III (e.g., Selye, 1976; Laughlin, 1967; Freud, 1895, 1917; Janet, 1900; Cannon, 1929; Fenichel, 1945).

To place these issues in a broader historical context, it is instructive to note that Sigmund Freud grappled extensively with the concept of PTSD (i.e., traumatic neurosis) from 1895 to 1920. In his book *Beyond the Pleasure Principle* (1920), he labored to distinguish between the core dynamics of traumatic neuroses and their relation to ego defense, anxiety, the concept of the stimulus barrier, and threat anticipation. In this work Freud elaborated on the idea that trauma could breach the stimulus barrier and overwhelm ego defenses, producing psychic trauma that could influence behavior, including manifestations of compulsions to repeat elements of the traumatic experience. Despite theoretical difficulties in resolving the differences between the traumatic neuroses, the war neuroses, and the anxiety-based neuroses, Freud understood that "mechanical violence of the trauma would liberate a *quantity* of 'sexual excitation' (i.e., stress response or pre-existing intrapsychic conflict) which, owing to the lack of preparation for anxiety, would have a traumatic effect" (Freud, 1920, p. 38; emphasis added). If a traumatic event had a *magnitude* of impact which overwhelmed coping resources, "the mechanism of the ego, including efforts to master the trauma in dream work, might not succeed" (Freud, 1920, p. 38). The potential for long-term stress effects on the dynamics of the psyche became obvious to Freud prior to World War II, long before the insertion of PTSD in the DSM-III (1980) diagnostic classification system. Moreover, as early as 1917 (i.e., during World War I), during his lectures in Vienna to the medical society, Freud identified and discussed *all* of the PTSD criteria that are listed in the current DSM-IV (1994); see his *Introductory Lectures on Psychoanalysis* (Freud, 1917), Chapter XVIII, "Fixation to Traumas—The Unconscious," for a detailed discussion of traumatic neuroses.

It is not our purpose in this introductory chapter to review the history and debate surrounding PTSD as a diagnostic entity, phenomenon, or process. Rather, it is to establish a framework in which to present the treatment goals for PTSD—a formidable and an extraordinarily interesting task, as will become evident in the chapters that follow. Clinicians, academics, and researchers face a multitude of considerations when attempting to understand and treat PTSD, as do patients trying to come to grips with and heal from its impact on their lives. As the book unfolds we hope that these factors will become evident and provide a sense of direction and understanding for practitioners, as well as for patients and their families affected by personal trauma.

Thoughtful examination will show that the complexity of the phenomena of PTSD will raise more questions than science can provide answers to at the present time, despite 16,925 articles in the international scientific database known by the acronym of PILOTS (Published International Literature on Traumatic Stress).[1] By the time this book reaches print, we expect that the worldwide database will contain between 18,000 and 20,000 annotated and indexed articles on the subject of traumatic stress and PTSD. So perhaps the most utilitarian, pragmatic, and scientific consequence of placing PTSD in the DSM-III of the American Psychiatric Association (1980) was that it opened the door to research scientists and other inquiring minds as to the nature, meaning, and structure of psychological trauma. What has happened since then speaks for itself in terms of scientific research, epidemiological studies, educational curricula and certification, the development of professional societies concerned with PTSD, and the urgency of understanding traumatic stress and PTSD in modern life.

We believe that it is important to clarify that posttraumatic phenomena are not limited to psychiatric diagnoses or decision making algorithms as defined in professional reference manuals (e.g., DSM-IV; American Psychiatric Association, 1994). PTSD symptoms listed in DSM-IV are primarily for use by clinicians who attempt to help their patients suffering from traumatic life experiences. We believe that it is necessary to expand these basic groupings of symptoms which define the *triad* of PTSD symptoms in order to maximize treatment effectiveness. Posttraumatic phenomena and their permutations are rich in their tapestry and are woven of thousands of threads whose fibers are spun from unique and sometimes exotic, secretive, horrific, and forbidden sources of discovery. Working clinically or in research settings with PTSD is a journey of puzzlement, curiosity, fascination, and uncertainty. At one end of the continuum, the work often exacts an enormous toll on therapists, draining their inner empathic resources (Dalenberg, 2000; Wilson & Lindy, 1994). At the other end of the continuum is the realization of the human capacity for resilience and self-actualization, and the power of the human spirit to heal itself. Practitioners working with PTSD clients often oscillate between the emotional extremes of this continuum. There is nothing

easy in their task; they often confront the worst horrors of human cruelty and malevolence. Listening to trauma stories is emotionally draining and hard work. As recent research has confirmed, clinical moments of dedication, inspiration, and hoped-for wisdom through education and training alternate with private reflections of self-doubt, insecurity, despair, and fantasies of escape from the heavy professional responsibility entailed in this task (Wilson & Thomas, 1999). Confronting human suffering through trauma work is often a painful process. It may result in professional burnout (Figley, 1995). The challenge and responsibility of the therapist who chooses to work with PTSD clients is to overcome burnout, empathic strain, maladaptive countertransference, and ineffective modalities of treatment. To do so demands endurance, commitment, and perseverance.

A second aim of this book is to identify areas of research, treatment, and clinical outcome which are not being addressed by the field of traumatic stress studies (Raphael & Wilson, 2000). This statement should not be construed as a criticism of the many excellent programs in the United States, Australia, Canada, Europe, Israel, and elsewhere where dedicated scholars labor to answer cutting-edge issues ranging from the epidemiological prevalence of PTSD (e.g., Kessler, 1995; Breslau, 1998) to the neuroscience of stress disorders (Bremner, 1999) to cross-cultural dynamics at work in processing psychological trauma (Kinzie, 1993).

In order to advance the field of studies in traumatic stress and PTSD, it is important to ask a series of critical questions. What are the voids in our knowledge base at this time? What fundamental sets of studies are necessary to define commonly agreed-upon advances in methodology, techniques of assessment, and neuroscience approaches? What are the consequences of traumatic experiences to epigenesis and life-stage development? What new educational endeavors need to be implemented in academic and professional training? What organizations (e.g., International Society for Traumatic Stress Studies [ISTSS]; National Center for Post-Traumatic Stress Disorders [NC-PTSD]; National Institute of Mental Health [NIMH]; United Nations International Children's Fund [UNICEF]; World Health Organization [WHO]; United Nations High Commission on Refugees [UNHCR]; International Critical Incident Stress Foundation [ICISF]; American Psychiatric Association; American Psychological Association; and American Academy of Experts in Trauma Studies [AAETS]) are going to undertake the responsibility of building bridges and foundations for cooperation, systematic planning, and program development in *all* of the areas which embrace and encompass the domains of trauma, stress disorders, and the myriad of related social-psychological and clinical phenomena?

As we enter the new millennium, how will such proactive programs be developed and facilitated in light of higher moral concerns for the future well-being of humankind and the quality of life worldwide? Dealing with human-induced traumas is a health-care priority as serious as any major

medical illness (U.S. Surgeon General, 1999). We believe that a broad and imaginative vision is critically needed if we are to advance to the further reaches of knowledge of the pathways to healing and recovery from PTSD and other related psychiatric phenomena (Maslow, 1971; Mack, 1999). With the acceleration of societal change brought on by the information age, we have seen that the new technologies are inducing rapid shifts in the patterns of day-to-day living and the rate at which ordinary people can access information on which to make decisions affecting their lives. In the field of traumatic stress studies, we must coordinate interorganizational/interagency cooperation to implement visionary agendas for the future and move in proactive ways beyond outdated models that limit innovative thinking and research.

When we consider the pressing issues which confront the field, it is evident that there is so much new ground to be unearthed and properly tilled that the task can sometimes seem daunting and even overwhelming. However, among the legacies of the 20th century is a "collective energy" to address these profoundly serious human concerns though many might wish to ignore them. Imagination, courage, risk taking, and the willingness to follow intuition are often accompanied by subjective feelings of danger and foreboding. As Abraham Maslow (1968) noted with brilliant lucidity, human growth motivation toward greater degrees of self-actualization enhances our attraction to the unknown, the uncertain, and less well-understood concepts in many areas of knowledge acquisition, especially as regards pressing contemporary issues. In contrast, fear, anxiety, insecurity, uncertainty, and the need for safety maximize our desire for the comfort of the known, the secure, and simple methods of plodding along, doing that which is conventional, unimaginative, and noncontroversial.

In the field of traumatic stress studies the current zeitgeist and momentum of the field impel us onward, realizing the humanitarian urgency of the task. Such a move toward greater scientific coordination and planning is a challenge and a mission. In this book we have chosen to take a small step in that direction and are hopeful that the issues presented by the contributors will stimulate new ways of thinking about treatment of trauma and PTSD, leading to healing. Our approach is conceptually holistic, dynamic, and rooted in the foundations of modern science, an approach driven by the marriage of theory, data, and clinical experience.

## THE SPECTRUM OF PTSD AND STRESS DISORDERS

The treatment approaches for PTSD recognize that it is a complex, dynamic entity rather than a unidimensional set of symptoms in a psychiatric reference manual. It is a premise of this book that PTSD represents a dramatic

and complex shift in the steady state of the organism. The concept of a spectrum of PTSD means that it can appear in different structural configurations. For example, the disorder may be expressed in a relatively "pure" sense of symptom presentation as defined in DSM-IV (1994). It may appear with other Axis I or Axis II disorders or be manifested as *complex PTSD*, with impacts on the inner core of the self-structure (Wilson & Zigelbaum, 1986; Marmar, Foy, Kazan, & Pynoos, 1993; Herman, 1992; Wilson, 1995). PTSD affects psychophysiological functioning in subtle and "masked" presentations, as illustrated by dissociative identity disorder (DID). The spectrum of PTSD is thus more than a diagnostic classification. There are relatively predictable forms of the disorder such as "pure" PTSD as a distinct, discrete anxiety phenomenon. There are also nonstatic, fluctuating PTSD states with extreme hyperarousal phenomena and complex defenses against underlying psychobiological processes (see Friedman, Chapter 4, this volume, and Wang, Wilson, & Mason, 1996). PTSD phenomena are manifested at multiple levels of synergistic processes: (1) stress-based emotional responses; (2) effects on cognitive-appraisal and information processing mechanisms; (3) psychobiological changes (i.e., neurohormonal), with feedback "loops" to organismic and system functioning (McEwen, 1998); (4) altered adaptation and coping behaviors; (5) effects on motives and goal-directed behaviors; and (6) shifts in spiritual and existential perspectives of day-to-day living and in the individual's sense of meaning and purpose (Wilson & Moran, 1997). The spectrum of PTSD therapy includes attempts to understand levels of consciousness and awareness (LCA) as part of the phenomenon itself. Most therapists who have experience in treating PTSD know that *unconscious* reenactment behaviors are not atypical (Blank, 1985; Wilson, 1989; Bremner & Marmar, 1998; Marmar, Weiss, & Metzler, 1997; Putnam, 1989). In terms of traumatic memories, van der Kolk (1999) and Goodwin (1993) described this phenomenon rudimentarily by the statement "emotional memories are forever." This research shows that traumatic residues exist within the memory bank of life experiences. As is well known, trauma can transform individual identity, the trajectory of the life cycle itself, and even subsequent generations (Wilson, 1980, 1988; Laufer, 1988; Danieli, 1994, 1998; Horowitz, 1999). Those who study trauma *ontologically* examine the vicissitudes of traumatic events and their transformation throughout life.

In Chapter 2, we present a holistic–dynamic model of PTSD and related psychological processes. We also present new models of PTSD as an allostatic organismic process (discussed further in the next section). These models are not only new forms of conceptualizing PTSD as a process but build on more than 20 years of accumulated scientific data. The "new paradigms" of emerging science and information utilization in the 21st century demand holistic, nonlinear models of complex stress-related phenomena. Among other historical legacies of the past century, PTSD has arisen rapidly to the attention of scientists and humanitarian workers because of its significance

for human evolution. Wars around the world and massive traumas such as the Holocaust and other genocidal outbreaks have increasingly threatened our existence as a species. Humankind may well not survive another century of annihilative conflicts employing ever-enhanced weapon systems of mass destruction. Paradoxically, the study of PTSD is ultimately about the need to find proactive mechanisms to eradicate those conditions which cause human sources of trauma in the first place (Wilson, 1995).

## TOWARD A NEW THEORETICAL PARADIGM OF PTSD

In the past few years, a series of research programs carried out by Bruce S. McEwen and his associates at Rockefeller University has explored the concept of allostasis and allostatic load in terms of the psychobiology of stress. Allostasis and allostatic load are related concepts and important to the understanding of PTSD and its treatment by one of the core treatment approaches. *Allostasis, unlike homeostasis, refers to the body's effort to maintain stability through change when loads or stressors of various types place demands on the normal levels of adaptive biological functioning.* According to McEwen (1998), allostasis is a response to the "wear and tear" that is produced by environmental demands (i.e., stressors of all types) which subsequently create allostatic loads—challenges to the system to maintain itself in a healthy and potentially optimal mode of functioning. The failure to "switch off" allostatic mechanisms once the threat or requirement to respond has terminated, however, begins a complex process of "wear and tear" on the nervous and hormonal systems.

As McEwen states (1998):

> The core of the body's response to challenge—whether it is a dangerous situation, an infection, living in a crowded and unpleasant neighborhood, or a public speaking test—*is twofold, turning on an allostasis response that initiates a complex adaptive pathway, and then shutting off this response[, which] involves the sympathetic nervous system and HPA [hypothalamic–pituitary–adrenal] axis.* For these symptoms, activation releases catecholamines from nerves and adrenal medulla and leads to secretion of corticotropism from the pituitary. . . . Inactivation returns the systems to baseline levels of cortisol and catecholamine secretion, which normally happens when the danger is past. . . . *However, if the inactivation is inefficient, there is over-exposure to stress hormones, over weeks, months or years, exposure to increased secretion of stress hormones can result in allostatic load and its pathophysiologic consequences.* (pp. 171–172, emphasis added)

The relevance of allostasis and allostatic load to PTSD phenomena is fundamental to the understanding of stress-related psychobiological behaviors. For many victims of trauma, the failure to resolve (i.e., integrate) the

traumatic experience within a new healthy baseline of normal psychobiological functioning renders them vulnerable in repeated ways to experience environmental cues (i.e., triggers) that can lead to a stable but abnormal adjustment characterized by intensification of the existing pathological stress responses which never fully terminated after the threatening (i.e., traumatic) situation ended. One of the major challenges of the core therapies for PTSD is to facilitate a reduction or "switching off" of *persistent hyperarousal mechanisms* associated with allostatic load that are readily reactivated and amplified by traumatic memories (conscious or unconscious) stored in the brain. Stated somewhat differently, persons suffering from PTSD are vulnerable to abrupt changes in their sense of well-being. They find themselves rapidly switching between states of relative calmness to states of hypervigilance, anxiety, anger, and extreme arousal. Sometimes the rapid switch is not readily understandable in terms of triggers or cues. As shown by van der Kolk (1999), Eitinger (1971), Freud (1917), and others, "the body keeps score." But unlike a baseball scoreboard where there are only two scores posted for each inning of play for the opposing teams, the body's "scoreboard" for allostasis in subtypes of PTSD is more like a powerful search engine of the most complex computer software in the organism's "internet" repertoire. Allostasis can affect virtually any domain of stored information and challenge the integrity of the system to execute its preprogrammed functions. When this occurs, a potential cascade of psychobiological processes can become "target" specific, as documented by Seeman and McEwen (1996) in their empirical study of health outcomes for subjects with higher versus lower degrees of allostatic load as operationally defined by psychobiological parameters (see Friedman, Chapter 4, this volume, for more detail).

Building on the seminal work of McEwen and his associates, we can apply the concept of allostasis and allostatic load directly to PTSD with specific implications for the core treatment approaches to PTSD. Initially, there is the normal, healthy response pattern to allostatic load: stress leads to coping and adaptation, followed by recovery and *homeostatic restability*. The healthy steady state is restored and continues in an optimal mode until called upon to respond again, with efficacy and mastery (White, 1959; McEwen, 1998; Antonovsky, 1979).

McEwen (1998) classifies four subtypes of allostatic load which produce "wear and tear" on the capacity to deal with stress, especially in PTSD because of the extreme nature of the traumatic stressor events. Briefly, these four patterns include the following: (1) *repeated hits* from multiple stressors in which the normal response pattern is frequently and repetitively activated, placing recurring demands on the system, which in turn tax effective coping; (2) the *lack of adaptation response* is similar to the above "repeated hits" subtype except that the effectiveness of normal adaptation starts to break down as the system's capacity to meet the load generated by the stressor is worn out, so that the system begins to fail at its genetically driven task; (3) the *prolonged stress*

*response,* in which the duration, frequency, or intensity of the traumatic event persists, as seen, for example, in war veterans, Holocaust survivors, political internees, and repeatedly abused children (Wang et al., 1996; Simpson, 1993; Pynoos & Nader, 1993; van der Kolk & Sapporta, 1993) (in these cases, the physiological response of allostatic adaptation continues, chronically activating the HPA axis [i.e., the biological stress response system] without relief and causing the stress hormones to persist in efforts to meet the ever-present demands of the stressors; in such cases, there may be no timely, proper, or adequate development and recovery period, thereby setting in motion *a synergistic pattern of pathological events in the brain and body*[2] that may have long-term deleterious consequences, some of which may become irreversible, permanent changes in both the structure and function of cortical, subcortical, and neurohormonal mechanisms [DeBellis et al., 1999]); (4) *inadequate response,* by which McEwen (1998) is referring to system failure, for example, the "inadequate secretion of glucocorticoids, resulting in increased concentrations of cytokines that are normally counter-regulated by glucocorticoids" (p. 174). McEwen argues convincingly that the various forms of allostatic load affect the brain and cardiovascular, metabolic, and immune systems.

When applied to the analysis of PTSD, we believe that it is possible to add a fifth subtype of allostatic load—the *combined-fusion model,* in which features of the other four subtypes coexist in relative degrees in different psychobiological systems: (1) repeated hits (multiple stressors); (2) lack of capacity for adaptation; (3) prolonged stress response; and (4) inadequate response, or system failure. Furthermore, they not only may exist in different degrees but may *alternate* with "rest" periods, even brief ones, of normal stress response periods, only to be followed by one or more of the allostatic load patterns. Wilson (1981, 1988) has clinically described this phenomenon in Vietnam combat veterans with heavy war zone exposure (i.e., prolonged stress response) and identified nine typologies of PTSD. For example, after repatriation, many Vietnam veterans had repeated problems of postwar adjustment, such as divorce, unemployment, substance abuse, social alienation, and loss of self-worth in society (i.e., repeated hits; multiple stressors), coupled with lack of adaptation due to inadequate stress response (i.e., system dysregulation, breakdown, and failure) (Lindy, 1986; Kulka et al., 1990). In such cases, the combined-fusion pattern of allostatic load led not only to "complex PTSD" but also to comorbidity (Lindy, 1986; Yehuda, 1998).

## PSYCHOLOGICAL THERAPIES FOR PTSD AND THE CRITERIA FOR RECOVERY, HEALING, AND REINTEGRATION OF THE SELF

What are the criteria by which to measure the healing and recovery from trauma? This question is germane to each of the treatment approaches out-

lined in this book. How is a specified treatment used to ameliorate allostatic load in PTSD? How is maximum stabilization achieved and the return toward optimum functioning restored to the individual? When does integration of the traumatic experience become a part of the general life perspective of the person rather than a fragmented, ego-alien, and unresolved bitter chapter in the life story (Horowitz, 1999)? How do therapists deal with persons who are so fragmented in their ego functioning that they have powerful unconscious self-destructive motives that subtly undermine the therapeutic process by attempting to re-create object-relational patterns which "justify" self-destructiveness, suicidality, and the malignant disruption of useful boundaries that have been established in therapy, friendships, family relationships, and the workplace (see Lindy & Wilson, Chapter 17, this volume, for a discussion)?

There can be no doubt that PTSD clients can create exceptionally difficult therapeutic relationships which engender powerful transference and countertransference relationships (Dalenberg, 2000; Wilson & Lindy, 1994). We believe that successful posttraumatic therapy (PTT) must know how to use the dynamics of the transference–countertransference matrix that exists in treatment settings in order to enter one of the five portals to the inner-core phenomena of PTSD which are the targets of treatment (see Wilson, Friedman, & Lindy, Chapter 2, this volume, regarding portals of entry for all domains of symptom treatment). It is our view that from a dynamic and holistic perspective the diversity and spectrum of PTSD typologies has three critical elements pertaining to the ego state of clients: (1) their perception of the trauma and its impact on their identity and personhood; (2) the allostatic disruption of their lives in terms of affect regulation and capacity to recognize and modify noneffective allostatic processes that perpetuate the syndrome rather than truncating nonadaptive stress response mechanisms; and (3) restoration of a meaningful sense of self-sameness and self-continuity (Erikson, 1968; Lifton, 1976, 1993; Wilson, 1989), which encompasses their view of themselves as persons having worth, dignity, wholeness, purpose, and an essential feeling of vitality. The healed self that was once traumatized can project itself into the future with joy, serenity, and a measure of wisdom. Persons who have transformed trauma can do so because of an awareness that the boundary separating the fear of threat from quiescence is more often than not illusory and only creates allostatic load when induced by cognitive appraisals of threat to the psychological basis of existence. The specter of loss of one's self through injury, or the death of a loved one can lead to a radical shift in the existential plane of beliefs and consciousness, as noted brilliantly by R. J. Lifton (1979), M. J. Horowitz (1999), and others in their pioneering contributions to the field. A shift in consciousness may lead to many different forms of behavior change, including a sense of spirituality. Writers of literature, many of whom endured war trauma, have given us poetry and fiction with new insights and sensitivity as to the frailty and re-

siliency of the human spirit. Psychotherapists and counselors use words such as "grounded," "centered," "integrated," "recovered," "healed," "transformed," "rejuvenated," "together," "transcended," "self-actualized," "psychosocially accelerated," and "spiritually connected" to characterize the extraordinary changes that occur when those afflicted by trauma emerge with a human radiance, energy, and dignity that is the total antitheses of illness, despair, suffering, and fragmentation of personality. Healthy and resilient survivors of trauma are persons who have found pathways to reverse or attenuate the destructiveness of psychic burdens which affect their health. They have freedom of consciousness to create active minds and bodies. They are also potential guides, healers, and teachers, and may be subjects of scientific inquiry concerning resiliency, salutogenesis, and self-efficacy. The study of healthy PTSD survivors (Krystal, 1968; Wilson, 1989; Antonovsky, 1968, 1979) ultimately may be more important than the study of those whose deterioration can only be stabilized or moderately reversed in the advanced stages of decompensation (Friedman, 2000; Wang, Wilson, & Mason, 1996).

The effort to find answers to questions of how recovery from PTSD occurs challenges those who are ready to move beyond the 20th-century models of trauma and coping dominated by psychopathology and illness (Wilson, Harel, & Kahana, 1988). Of course, understanding stress disorders remains of critical importance, but expanding our knowledge of regenerative health and vitality is now an imperative in an era of innovations in humankind's capacity to shape itself in ways never before imagined.

Transforming the psychobiological expressions of stress-related illness and enlarging our capacity to restore the well-being of clients are tangible possibilities. Traumatic and untreated stress, in the broadest medicopsychological sense, can cause (1) physical illness, (2) the loss of self-realization or growth, (3) and a disruption of the life-course trajectory. The core therapeutic approaches to PTSD seek inroads to facilitate innovative and effective modalities of healing traumatic injury. We suggest that a transformation of consciousness can be a key part of PTSD therapy (Wilson, 1980; Wilson & Moran, 1997).

## THE SCIENTIST-PRACTITIONER: CRITERIA AND STANDARDS FOR DEFINING THE SUCCESSFUL TREATMENT OF PTSD

It has been traditional in the history of psychotherapy, especially in debates surrounding the most effective approach to helping clients with PTSD, to argue as to what "works best" in alleviating symptoms (Nathan & Gorman, 1998). On the one hand, there are the pragmatists who take the view that if a clinical technique "works" to produce the relief of symptoms, then its use

and practice is justified, especially if clients report that they "feel better" (Williams & Sommer, 1994). On the other hand, there are the "hardheaded" researchers who demand technical–scientific proof of therapeutic efficacy through controlled and repeated clinical trials which are subject to the most rigorous and conservative standards of modern research methodology (Foa & Meadows, 1997). These opposing views are readily appreciated and understood because they reflect different professional roles and responsibilities, despite the fact that both positions are committed to the ethical principles of "doing no harm" to the patient and upholding the highest standards of practice. However, when it comes to the treatment for PTSD, we must move toward a synthesis of the two divergent and well-justified approaches.

Foa and Meadows (1997), in the *Annual Review of Psychology*, Volume 48, argue that there are "Gold Standards" by which to determine treatment outcome studies of PTSD. They suggest seven general methodological procedures: (1) *clearly defined target symptoms* (e.g., distressing intrusive recollections—traumatic memories; (2) the use of *reliable and valid measures*; (3) use of *blind evaluations* (i.e., independent raters with no biases) in measuring symptom improvement; (4) *assessor training*, which includes such things as interrelator reliability and familiarity with the clinical syndrome; (5) *manualized, replicable treatment programs* (i.e., structural, standardized protocols); (6) *unbiased assignment to treatment* (i.e., the use of randomization); and (7) *treatment adherence* (i.e., monitoring compliance with the treatment program being used).

To begin, it is useful to specify some of the areas in which the objectives of successful treatment of PTSD are in concurrence in the clinical and scientific literature. Our approach builds on the model of allostasis and allostatic load in the subtypes of PTSD (discussed earlier), woven within a theoretical fabric of a holistic–dynamic approach to the treatment of PTSD.

## Objectives of the Treatment Approaches for PTSD

In the simplest formulation, the central objectives in the treatment of PTSD are as follows: (1) normalization of the stress response, that is, attenuate allostatic load and allostatic processes that perpetuate maladaptive and prolonged psychobiological stress responses within the organism to alleviate anxiety, tension, and levels of distress; (2) facilitate a reduction or elimination of maladaptive psychobiological processes which include cognitive distortion, hyperarousal processes, hypervigilance, startle responses, sleep disturbance, and affective instability ranging on a continuum from anger to depression to diverse forms of anxiety. In terms of anxiety management, Keane (1998) and Foa and Meadows (1997) (see also Zoellner, Fitzgibbons, & Foa, Chapter 7, this volume) have reviewed the various techniques for clinically managing the anxiety spectrum of PTSD, including cognitive-behavioral treatments (CBT), exposure procedures (EP), *in vivo* exposure procedures (VP), anxiety management treatment (AMT) programs, and stress inoculation training

(SIT). In their 1997 summary based on a review of the literature, Foa and Meadows concluded:

> Overall, cognitive-behavioral treatments enjoy the greatest number of controlled outcome studies, and have been the most rigorously tested. Those studies converge to demonstrate that both prolonged exposure procedures and stress inoculation training are effective in reducing symptoms of PTSD. CPT (cognitive processing treatment) has shown promising initial findings, but it awaits the results of more rigorously controlled studies before its efficacy can be determined. (p. 474)

Keane (1998) reaches virtually the same conclusion in his review, suggesting that there is a concurrence of information pointing toward the conclusion proposed above that reductions in allostatic load has generalizable effects in the psychobiologically based dimensions of the anxiety–depression–hyperarousal spectrum.

The various techniques (CBT, AMT, PE, etc.) that have shown effectiveness in treating the salient symptoms of PTSD, measured by different techniques (see Foa & Meadows, 1997), are consistent with Friedman's view (see Chapter 4, this volume) of PTSD as a psychobiological state. The use of the term "psychobiological" is important since there is no dualism being proposed between mind and body. Stated simply, *allostatic processes are inextricably linked to the spectrum of PTSD phenomena.* The therapeutic technologies reviewed by Foa and Meadows (1997) may be effective for the anxiety-based dimensions of PTSD, but are they sufficient for other aspects of the disorder, such as the client's impaired sense of integrity, wholeness, self-esteem, and personal identity, as well as his or her proneness to dissociation and high-risk-taking behaviors?

Viewed from a different perspective, what treatments work best for which kind of PTSD client and under what circumstances? Table 1.1 illustrates this relationship and is particularly important when therapists are considering the use of any of the core treatment approaches for PTSD.

## PTSD as a Psychobiological Stress Response Syndrome: Implications for Treatment

Serving as a brief summary, Table 1.1 encapsulates how the subtypes of allostatic load are associated with PTSD processes. In Chapter 4 of this volume, Friedman expands upon these psychobiological mechanisms and related issues in greater detail, considering their many implications for treatment.

1. *Altered thresholds of response.* Allostasis implies that there are degrees of altered thresholds of response. Behaviorally, these include the degrees of readiness to respond, levels of hyperarousal, and altered appraisal processes,

**TABLE 1.1. PTSD as Psychobiological Allostasis: Treatment Implications**

| Allostatic process | Associated PTSD symptoms | DSM-IV PTSD criteria |
|---|---|---|
| 1. Altered *threshold* of response | Readiness to respond; hypervigilance; altered appraisal processes; increased threat appraisal; proneness to reenactment or reexperience; lower stress tolerance | B1, B3, B4, B5, C1, C2, D1, D2, D3, D4, D5 |
| 2. *Hyperreactivity*: allostatic dysregulation | Irritability; proneness to aggression; physiological and psychobiological hyperreactivity; startle response; insomnia; avoidance tendencies; inability to modulate arousal and affect | B1, B3, B5, D1, D2, D3, D4, D5 |
| 3. Altered *initial* response patterns | Decreased safety appraisal; decreased stress tolerance; overreaction to external or internal cues; proneness to fight-or-flight response | B3, B4, B5, C1, C2, D2, D4 |
| 4. Altered *capacity* of internal monitoring | Decreased capacity for accurate self-monitoring; increased vulnerability of cognitive and emotional response | B3, B4, B5, C6, D4 |
| 5. Altered *feedback* based on distorted information | Decreased capacity for accurate monitoring of interpersonal events and effects on others; altered cognitive schemas; erroneous cognitions of self and world | B3, C1, C2, C3, C5, C6, D2, D4 |
| 6. Altered *continuous* response | Increased proneness to avoidance and dissociation, amnesia, hyperarousal, cognitive dysregulations and somatic expressions of distress; insomnia; startle response | B1, B2, B3, B4, B5, C1, C2, C3, D1, D4, D5 |
| 7. *Failure to habituate*: failure of system to "shut down" and restore homeostasis (i.e., allostatic load) | Increased proneness to reenactment, traumatic memory, fluctuating levels of arousal; proneness to act out and reenact posttraumatic events; sleep disturbance; avoidance patterns; startle response | B1, B3, B4, B5 C1, C2, D1, D2 D4, D5 |
| 8. Establishment of new level of allostatic steady-state adaptations | Encompasses all of the above. (1–7) | All B, C, D |

especially threat appraisals. The perception and appraisal of threat is *trauma specific in nature* (Wilson & Lindy, 1994; Dalenberg, 2000). Thus, depending on the particular event witnessed, endured, or survived, a PTSD client will have different sensitivity thresholds and memories as to cues associated with the appraisal process and its implication for thresholds of behavioral responsiveness in allostatic mechanisms.

2. *Hyperreactivity: Allostatic dysregulation.* Hyperreactivity is one component of the psychobiology of PTSD. Hyperreactivity refers to allostatic dysregulation and is associated with an inability to modulate arousal and affect. This lack of capacity for regulating arousal and affect is associated with irritability, proneness to aggression, exaggerated startle response, insomnia, hypervigilance, and excessive autonomic nervous system arousal. Persons prone to modes of hyperreactivity in PTSD may alternate between displays of threat, aggression, and intimidation, on the one hand, and isolation, detachment, and withdrawal from others, on the other. In either mode, there is a behavioral attempt to impose structure and control which is missing in situations due to dysregulation. Prolonged states of hyperreactivity may lead to fatigue, exhaustion, and depressive symptoms (i.e., hypersomnia, loss of initiative and striving, weight loss or gain, feelings of being "blue" and "down in the dumps," and the like). Clinically, persons suffering from high levels of hyperreactivity behaviors may be misdiagnosed as having bipolar disorder because states of high arousal and energy may appear manic-like and, when fatigue occurs leading to detachment, withdrawal, and isolation, may manifest a depressed-like state in demeanor and affect.

3. *Altered initial response thresholds.* Allostatic loads influence the predisposition to initial response patterns in PTSD. This includes such examples as decreased capacity for accurate self-monitoring of emotional states (e.g., anger, psychic numbing, affective constriction or effects of alcohol consumption). More essential is that altered response threshold as disposition is experienced as subjective vulnerability, which in turn affects cognitive appraisals, ego defensiveness, and readiness to respond to cognitive appraisals. As will be discussed further by Lindy and Wilson (see Chapter 17, this volume), ego vulnerability is at the core of the most severe and radical of PTSD disturbance. But what constitutes "vulnerability" is a complex question compounded by genetics, personality, and trauma-based experiences. *However, once situated within the personality, the individual's subjective perception of personal vulnerability has enormous implications for cognitive schemas, especially threat appraisal and risk-taking behaviors* (Wilson, 1989; Aronoff & Wilson, 1985; Krystal, 1968; Lifton, 1993; Dalenberg, 2000).

4. *Altered capacity of internal monitoring.* Another allostatic process common to PTSD is an altered capacity to monitor internal states. This refers to a decreased capacity to accurately "read" (i.e., self-monitor) levels of hyperarousal as well as affective states. The inability to monitor and experience af-

fective states includes degrees of psychic numbing, emotional blunting, or anesthesia in which feelings are absent or inaccessible to individual perception and recognition. Moreover, the altered capacity for self-monitoring has implications for cognitive processing, interpersonal relations, and subjectively experienced states of vulnerability. The failure to accurately monitor and process internal states creates the possibility for misperceiving others' intentions and emotional states by cognitive distortion or simply as failure to feel empathically their emotional state of being. In PTSD ego states increased vulnerability occurs because a loss of capacity for internal monitoring results in faulty information processing and "signal" detection from cues in others and the environment. When the capacity to adequately monitor internal states leads to faulty, distorted, or inadequate person perception or situational cue analysis, a heightened sense of vulnerability may result. As discussed by Lindy and Wilson in Chapter 5 of this volume, increased vulnerability leads to defensive adaptations to ward off anxiety, fear and uncertainty.

5. *Altered feedback based on distorted information.* Allostatic load is associated with the phenomena described in the last section, but also includes cognitive alterations in schemas. Elsewhere, Wilson (1989) identified five common subtypes of cognitive alterations in response to traumatic stressors: (a) *denial/avoidance* of the stressor or stressors as events or specific stimulus cues; (b) *cognitive and/or perceptual distortions* (e.g., augmentation or reduction of a perceptual modality—visual, auditory, olfactory, or kinesthetic); (c) *accurate appraisal of the traumatic events*; (d) *dissociation* (e.g., derealization, depersonalization, or amnesia); and (e) the *peritraumatic onset of memories* associated with the event itself—in other words immediate, intrusive recollections of what just took place in the traumatic situation (Bremner & Marmar, 1998; Singer, 1990; Cohen, Lewis, Berzoff, & Elin, 1997).

6. *Altered Continuous Responding.* Allostatic load has also been associated with the consequence of increasing proneness to dissociation (due to system overload in information processing) in any of its well researched forms (see Steinberg, 1997, for a review). Further, altered continuous responding is related to the threshold of responsiveness of behavioral adaptation. Hence, hypervigilance and alterations or transformations in cognitive processes (i.e., memory, problem solving, executive functioning, data interpretation, and categorization of newly acquired information, etc.) are but a few examples of how cognitive-perceptual and motivational dimensions of PTSD can combine in complex psycho-algorithmic formulas to affect allostatic processes.

7. *Altered continuous response.* Altered continuous response is another form of allostatic processes. In this process, the continuous flow of behavior, coping, and adaptation is disrupted. Disrupted response tendencies are manifest in psychobiological ways which include emotional lability and distress, somatic expressions (e.g., fatigue, headaches, bodily complaints or sleep disturbance), exaggerated startle response, and hyperaroused states. Furthermore, other forms of altered continuous response patterns may be seen in dissocia-

tion (i.e., altering conscious mental activity), amnesias, increased proneness to avoidance (e.g., geographic isolation, emotional detachment, and/or social noninvolvement with others) Finally, altered cognitive processes, such as information processing, attention, memory, and higher-order executive functions, may be expressed allostatically as well.

8. *Nonhabituation: The failure of the allostatic system to "shut down" and restore homeostasis.* The presence of allostatic load drives the entire autonomic nervous system and related endocrine functions to varying degrees. By this we mean that allostatic load can have a profound impact on the HPA axis, as noted by McEwen (1998) and Friedman (1990), but eventually have systemic effects as well, often to organ systems. The failure of the system to shut down thus increases the full spectrum of PTSD behaviors from reenactment phenomena to fluctuating levels of hyperarousal, which may alternate in affective manifestations in varying combinations (e.g., hyperarousal → depression → anxiety → anger → withdrawal or acting out inner tensions). Thus, the failure to habituate encompasses all other forms of allostatic processes but must be categorized separately because it reflects what McEwen termed inadequate response to return to homeostasis. The failure to habituate implies much more than the original stress formulation proposed in the brilliant early work of Hans Selye (1976), namely, alarm reaction (A), resistance (R), and exhaustion (E). The general adaptation syndrome (GAS = A, R, E) described by Selye is both organ specific and cognitive in nature. Indeed, the GAS is one of the earliest formulations of the process of allostasis and its effects within the organism. However, Selye considered the GAS as nonspecific responses to stressors, whereas allostasis specifies the pathways of disturbed functions caused by system overload.

## ALLOSTATIC TRANSFORMATIONS IN PTSD

As Freud noted in *Beyond the Pleasure Principle* (1920), the breach of the stimulus barrier may lead to "hypercathexis" and other consequences delineated in psychoanalytic terminology (Lindy, 1993; Wilson & Lindy, 1994). The failure to return to equilibrium or homeostatic states due to allostatic load has two basic principles which are the psychobiological "brick and mortar" of the stress syndrome: (1) *lowered stress tolerance,* which may trigger a cascade of PTSD phenomena; and (2) *the psychobiological memory of trauma,* which produces behavioral states of overreadiness to respond to situations due to hyperarousal, hypervigilance, decreased accurate self and other-monitoring and cognitive dysregulations in memory, thinking, information processing, judgment, perception, and appraisal processes, especially those of perceived threat. Lowered stress tolerance renders the trauma client even more vulnerable; a wider range of stimuli may act as triggers or cues evoking one or more of the syndrome dynamics outlined in Table 1.1. From a psychodynamic per-

spective, this makes the human psyche even more complicated because each of the forms of PTSD as allostatic transformations has the potential to interact and intensify, augment (amplify), or attenuate one aspect of the system. As discussed later in this chapter, relational patterns, ranging from total isolation to active group membership, may play a significant role in recovery and restoration of the self. *Healthy recovery involves the capacity to find a role in a significant group or society that allows a sense of personal integrity without the loss of selfhood and self-fragmentation, as well as the ability to sustain commitments and responsibilities that define the survivor's continuity of daily life.*

## PSYCHOLOGICAL TREATMENTS FOR PTSD

It is one of the primary objectives of this book to present the treatment approaches for PTSD and to do so within a holistic–dynamic theoretical perspective. To provide adequate care for someone suffering from PTSD requires an understanding of the dynamics and complexity of the phenomenon (Matsakis, 1994). In this final section, we present a framework of the internal and external manifestations of the stress disorder.

As noted by Friedman (2000) the exponential growth of research on PTSD has enabled educators, consumers, scientific researchers, and others to select from a fairly vast array of information in the following areas most relevant to the treatment approaches for PTSD: (1) diagnostic criteria; (2) psychological assessment and clinical interview procedures; (3) differential diagnoses (i.e., taking into account how PTSD is similar to or different from other psychological disorders); (4) the various treatment options available; (5) specialized treatments for children; and (6) medical and pharmacological options that are available, ranging from medications to inpatient treatment programs.

## TREATMENT GOALS FOR TRAUMATIC STRESS SYNDROMES

What are the common treatment goals for PTSD? Marmar et al. (1993), in the *Review of Psychiatry* (Volume 12), presented one of the first attempts to present "an integrated approach for treating post-traumatic stress" (pp. 239–272). Among the important contributions of their review was the identification of 13 common treatment goals by psychodynamic, cognitive-behavioral, and pharmacological approaches. These treatment goals were more or less universal in nature; that is, they apply to simple and complex forms of PTSD which correspond remarkably closely to McEwen's subtypes of allostatic processes. Marmar et al. (1993) classified traumatic stress categories into five groupings: (1) normal stress response; (2) acute catastrophic stress re-

action; (3) PTSD without comorbidity; (4) PTSD with Axis I comorbidity; and (5) PTSD with Axis II comorbidity. In their review, the authors discuss the application of the three types of psychotherapies (i.e., psychodynamic, cognitive-behavioral, and pharmacological) to these five categories of traumatic stress. Thus, despite the type of traumatic event or psychotherapeutic approach, the 13 common treatment goals share common objectives in terms of reducing allostatic load. Included in the list of the 13 treatment objectives are such factors as reduced levels of hyperarousal, accurate threat appraisal, return to the normal pathway of psychosocial development, reduction in traumatic memories; reduction of comorbid problems, restoration of integrity and self-esteem, and education about the stress process associated with PTSD as a disorder.

Table 1.2 presents a summary of the five core areas of PTSD and the general treatment goals for them. Later, in Chapters 2 and 3, we discuss these areas in much greater detail from a psychodynamic and psychobiological perspective. Moreover, each of the individual chapters on the core treatment approaches presents even more detail and discussion than space permits here. A general summary is useful as an introduction to the later chapters.

Each domain of PTSD has a set of target objectives which Wilson discusses in Chapter 3 of this volume in greater detail. Here, however, we can identify treatment goals by the symptom clusters. First, in terms of psychobiological alterations, the two primary goals are (1) to reestablish the normal (healthy) stress response to the extent possible and (2) to identify allostatic changes such as sleep disturbances, hypervigilance, irritability, proneness to anger, problems of concentration, and vulnerability to medical illness.

In terms of traumatic memory, the treatment goals from an allostatic perspective include identifying triggers for intrusive, distressing recollections of the trauma; uncoupling traumatic memory from debilitating affects and gaining authority and mastery over anxiety-provoking processes through cognitive reappraisal mechanisms and desensitization procedures.

The treatment goals for the avoidance, numbing, and denial cluster are primarily centered around the development of insight into maladaptive avoidance activities as part of PTSD and learning positive coping skills of various sorts that increase a sense of self-control, autonomy, and capacity for healthy self-esteem in the day-to-day transactions of living.

When traumatic events produce damage to ego processes (i.e., the self-structure, personal identity, and adequacy of the self-concept) the treatment goals are to reduce narcissistic injury to the self and to promote the integration of the traumatic experience within the self-schema of the individual so that it is not experienced as ego alien but as part of the life-history of the person. The primary treatment goals for this cluster of symptoms includes correcting faulty cognitions about self and world and gaining insight into states of experienced vulnerability and the use of ego-defense mechanisms to protect areas of injury to the coherency to the self-structure.

**TABLE 1.2. Common Goals in Treatment for PTSD and Their Relation to Allostasis**

| PTSD dimension | PTSD treatment goal |
|---|---|
| 1. *Psychobiological alterations:* hypervigilance, irritability, proneness to anger, depression, emotional lability, exaggerated startle response, sleep disturbance, problems of concentration, dissociation, somatic expressions of PTSD, vulnerability to medical illness | Reestablish normal stress response; normalize PTSD as psychobiological process; medicate as necessary; restore sleep and relaxation mechanisms (e.g., biofeedback, exercise); understand dissociation as hyperarousal; gain insight into medical/somatic expressions of PTSD |
| 2. *Traumatic memory:* intrusive recollections, nightmares, emotional (somatic) memories, acting-out/reliving trauma, reenactment play, perceptual illusions, dissociation, memory retrieval | Identify "triggers" for memories; active cognitive reappraisal; understand dissociative episodes; integrate memories of trauma; uncouple memory from debilitating affect; gain mastery over fear and distress; learn accurate appraisal of anxiety and threat stimuli |
| 3. *Avoidance, numbing, and denial:* avoidance, emotional constriction/numbing, amnesia, loss of active social interpersonal engagement, substance abuse, social/geographic isolation, desexualization, estrangement and detachment, obsessive–compulsive, attention diversion as defense | Gain insight into development and use of avoidance/numbing/denial mechanisms; facilitate recall of fragmented, amnestic, repressed, or blocked memories; treat substance abuse concurrent with PTSD; restore self-esteem and identity as survivor rather than victim; find ways to reconnect to self, others, and meaningful activities; reduce maladaptive coping behavior; uncouple memory from fear response |
| 4. *Self-concept, ego states, personal identity, and self-structure:* demoralization, ego fragmentation, identity diffusion, proneness to dissociation, hopelessness and helplessness, vulnerability, loss of spirit and vitality, dysphoria, shame, guilt, misanthropic beliefs, faulty cognitions about self and world | Reduce narcissistic injury to self; restore self-esteem; restore personal integrity and vitality; decrease sense of vulnerability; integrate trauma experience in self-concept; identify risks of suicidality; place trauma within developmental perspective; facilitate normal psychosocial development and understand changes in life-course developmental trajectory; correct faulty cognitions about self and world |
| 5. *Attachment, intimacy, and interpersonal relations:* Alienation; mistrust, detachment; self-destructive relationships; somatic tension; "boundary" problems with others; issues of loss, abandonment, impulsiveness, and object relations deficits | Restore good personal relations; learn to establish healthy boundaries; confront emotional feelings associated with vulnerability, detachments, and problem areas associated with bodily tension, invoke self-trust and capacity for meaningful personal relationships |

PTSD includes, in many cases, impacts on attachment behaviors, capacity for intimate relations, and the quality of interpersonal encounters. Treatment objectives for this domain of symptoms includes learning to establish or maintain boundaries; reducing alienation, isolation, detachment, mistrust, and self-defeating behaviors. In order to restore or maintain good personal relations, it is necessary for the client to understand the connection between vulnerability states (e.g., fears, feelings, perceived threats) and dispositional tendencies in social encounters. Clearly, the link between *intra*personal dynamics in ego states and *inter*personal dynamics is an important one, and treatment goals should attempt to identify the manner in which they influence each other in reciprocal ways.

As a general summary, Table 1.2 presents the common goals for the treatment of PTSD symptoms classified according to the five domains which constitute the disorder viewed as an allostatic process.

## NOTES

1. Personal correspondence with Dr. Fred Lener, November 30, 1999, National Center for PTSD, White River Junction, VT. The PILOTS database is available on-line at:

*www.ncptsd.org*

2. This might be construed as fatigue or somatic weariness, which has been referred to often in the PTSD literature (Wilson & Raphael, 1993).

## REFERENCES

American Psychiatric Association. (1952). *Diagnostic and statistical manual of mental disorders* (1st ed.). Washington, DC: Author.

American Psychiatric Association. (1968). *Diagnostic and statistical manual of mental disorders* (2nd ed.). Washington, DC: Author.

American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, DC: Author.

American Psychiatric Association. (1987). *Diagnostic and statistical manual of mental disorders* (3rd ed., rev.). Washington, DC: Author.

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

Antonovsky, A. (1968). Social class and the major cardiovascular diseases. *Journal of Chronic Diseases, 21,* 65–106.

Antonovsky, A. (1979). *Health, stress and coping.* San Francisco: Jossey-Bass.

Aronoff, J., & Wilson, J. P. (1985). *Personality in the social process.* Hillsdale, NJ: Erlbaum.

Blank, A. S., Jr. (1985). Irrational reactions to posttraumatic stress disorder and Vietnam veterans. In S. M. Sonnenberg, A. S. Blank, Jr., & J. A. Talbott (Eds.), *The*

*trauma of war: Stress and recovery in Vietnam veterans* (pp. 21–34). Washington, DC: American Psychiatric Press.

Bremner, J. D. (1999). Alternations in brain structure and function associated with posttraumatic stress disorder. *Seminars in Clinical Neuropsychiatry, 4,* 249–255.

Bremner, J. D., & Marmar, C. R. (1998). *Dissociation.* Washington, DC: American Psychiatric Press.

Breslau, N. (1998). Epidemiology of trauma and posttraumatic stress disorder. In R. Yehuda (Ed.), *Psychological trauma* (pp. 1–27). Washington, DC: American Psychiatric Press.

Breslau, N., & Davis, G. C. (1992). Posttraumatic stress disorder in urban populations of young: Risk factors in for chronicity. *American Journal of Psychiatry, 149,* 671–675.

Cannon, W. B. (1929). *Bodily changes in fear, hunger, pain and rage.* New York: Appleton-Century.

Cohen, S., Lewis, M., Berzoff, J. N., & Elin, M. R. (1997). *Dissociative identity disorder: Theoretical and treatment controversies.* Northvale, NJ: Aronson.

Dalenberg, C. L. (2000). *Countertransference in the treatment of trauma.* Washington, DC: American Psychological Association Press.

Danieli, Y. (1994). Countertransference, trauma, and training. In J. P. Wilson & T. J. Keane (Eds.), *Assessing psychological trauma and PTSD* (pp. 368–389). Westport, CT: Greenwood Press/Praeger.

Danieli, Y. (Ed.) (1998). *International handbook of multigenerational legacies of trauma.* New York: Plenum Press.

DeBellis, J. M., Baum, A. S., Birnaber, B., Kesharan, M. S., Eccard, C. H., Boring, A., Jenkins, F. J., & Ryan, N. (1999). Developmental traumatology, Part I: Biological stress systems. *Biological Psychiatry, 45,* 1259–1270.

Eitinger, L. (1980). The concentration camp syndrome and its late sequela. In J. E. Dimsdale (Ed.), *Survivors, victims, and perpetrators: Essays on the Holocaust.* New York: Hemisphere Press.

Erikson, E. (1968). *Identity, youth and crisis.* New York: Norton.

Fenichel, O. (1945). *Psychoanalytic theory of neurosis.* New York.: W.W. Norton.

Figley, C. (1995). *Compassion fatigue.* New York: Brunner/Mazel.

Foa, E. B., & Meadows, E. A. (1997). Psychosocial treatments for posttraumatic stress disorder: A critical review. In J. T. Spence, J. M. Dorley, & D. J. Foss (Eds.), *Annual review of psychology* (Vol. 48, pp. 449–480). Palo Alto, CA: Annual Reviews.

Freud, S., & Brewer, J. (1895). *Studies on hysteria.* New York: Avon Press.

Freud, S. (1917). *Introductory lectures on psychoanalysis* (J. Strachey, Trans.). New York: Norton.

Freud, S. (1920). *Beyond the pleasure principle* (J. Strachey, Trans.). New York: W.W. Norton.

Friedman, M. J. (1990). Interrelationships between biological mechanisms and pharmacotherapy of post-traumatic stress disorder. In M. E. Wolfe & A. D. Mosnian (Eds.), *Post-traumatic stress disorder: Etiology, phenomenology and treatment* (pp. 204–205). Washington, DC: American Psychiatric Press.

Friedman, M. J. (2000). *Post-traumatic and acute stress disorders.* Kansas City, MO: Compact Clinicals.

Goodwin, J. M. (1993). *Rediscovering childhood trauma.* Washington, DC: American Psychiatric Press.

Herman, J. (1992). *Trauma and recovery.* New York: Basic Books.

Horowitz, M. J. (1986). *Stress response syndromes.* New York: Aronson.

Horowitz, M. J. (1999). *Essential papers on post-traumatic stress disorder.* New York: New York University Press.

Janet, P. (1900). *L'automatismre psychologique.* Paris: Ballière.

Keane, T. M. (1998). Psychological and behavioral treatments for post-traumatic stress disorder. In P. E. Nathan & J. M. Gorman (Eds.), *Treatments that work* (pp. 398–408). New York: Oxford University Press.

Kessler, N. C., Sonnega, A., & Bromet, E. (1995). Post-traumatic stress disorder in the national co-morbidity survey. *Archives—General Psychiatry, 52,* 1048–1060.

Kinzie, J. D. (1993). Posttraumatic effects and their treatment among Southeast Asian refugees. In J. P. Wilson & B. Raphael (Eds.), *International handbook of traumatic stress syndromes* (pp. 311–321). New York: Plenum Press.

Krystal, H. (1968). *Massive psychic trauma.* New York: International Universities Press.

Kulka, R., Schlenger, W., Fairbank, J., Hough, R., Jordan, B. D., Marmar, C. R., & Weiss, D. (1990). *Trauma and the Vietnam war generation.* New York: Brunner/Mazel.

Laufer, R. S. (1988). The serial self: War trauma, identity and adult development. In J. P. Wilson, Z. Harel, & B. Kahane (Eds.), *Human adaptation to extreme stress: From the Holocaust to Vietnam* (pp. 33–53). New York: Plenum Press.

Laughlin, H. (1967). *The neuroses.* Washington, DC: Butterworth.

Lifton, R. J. (1976). *The life of the self.* New York: Simon & Schuster.

Lifton, R. J. (1979). *The broken connection: On death and the continuity of life.* New York: Basic Books.

Lifton, R. J. (1993). From Hiroshima to the Nazi doctors: The evaluation of psychoformative approaches to understanding traumatic stress syndromes. In J. P. Wilson & B. Raphael (Eds.), *International handbook of traumatic stress syndromes* (pp. 11–25). New York: Plenum Press.

Lindy, J. D. (1986). *Vietnam: A carebook.* New York: Brunner/Mazel.

Lindy, J. (1993). Focal psychoanalytical psychotherapy of post-traumatic stress disorder. In J. P. Wilson & B. Raphael (Eds.), *International handbook of traumatic stress syndrome* (pp. 803–811). New York: Plenum Press.

Mack, J. D. (1999). *Passport to the cosmos.* New York: Crown Books.

Marmar, C. R., Foy, D., Kazan, B., & Pynoos, R. S. (1993). *An integrated approach for treating posttraumatic stress.* In T. M. Oldham, M. B. Riba, & A. Tasman (Eds.), *Review of psychiatry* (Vol. 12, pp. 239–273). Washington, DC: American Psychiatric Press.

Marmar, C. R., Weiss, D. S., & Metzler, T. M. (1997). The Peritraumatic Dissociative Experiences Questionnaire. In J. P. Wilson & T. M. Keane (Eds.), *Assessing psychological trauma and PTSD* (pp. 412–429). New York: Guilford Press.

Maslow, A. H. (1968). *Towards a psychology of being.* New York: Van Nostrand.

Maslow, A. H. (1971). *The farther reaches of human nature.* New York: Viking Press.

Matsakis, A. (1994). *Post-traumatic stress disorder.* Oakland, CA: New Harbinger.

McEwen, B. S. (1998). Protective and damaging effects of stress mediators. *Seminars in Medicine of the Beth Israel Deaconess Medical Center, 338*(3), 171–179.

Nathan, P. E., & Gorman, J. M. (1998). *Treatments that work.* New York: Oxford University Press.

Putnam, F. W. (1989). *Diagnosis and treatment of multiple personality disorder.* New York: Guilford Press.

Pynoos, R., & Nader, K. (1993). Issues in the treatment of posttraumatic stress in children. In J. P. Wilson & B. Raphael (Eds.), *International handbook of traumatic stress syndromes* (pp. 527–535). New York: Plenum Press.

Raphael, B., & Wilson, J. P. (2000). *Psychological debriefing: Theory, practice, evidence.* Cambridge, UK: Cambridge University Press.

Seeman, T. E., & McEwen, B. S. (1996). The impact of social environment characteristics on neuroendocrine regulations. *Psychosomatic Medicine, 58,* 459–471.

Selye, H. (1976). *The stress of life.* New York: McGraw-Hill.

Simpson, M. (1993). Traumatic stress and the bruising of the soul: The effects of torture and coercive interrogation. In J. P. Wilson & B. Raphael (Eds.), *International handbook of traumatic stress syndromes* (pp. 659–667). New York: Plenum Press.

Singer, J. L. (Ed.). (1990). *Repression and dissociation: Implications for personality theory, psychopathology and health.* Chicago: University of Chicago Press.

Steinberg, M. (1997). Assessing posttraumatic dissociation with structural clinical interviews for DSM-IV dissociative disorders. In J. P. Wilson & T. M. Keane (Eds.), *Assessing psychological trauma and PTSD* (pp. 429–448). New York: Guilford Press.

U.S. Surgeon General. (1999, December). *Mental health: A report of the Surgeon General.* Washington, DC: U.S. Public Health Service, Department of Health & Human Services.

van der Kolk, B. (1999). The body keeps score: Memory and the evolving psychobiology of posttraumatic stress. In M. Horowitz (Ed.), *Essential papers on posttraumatic stress disorder* (pp. 301–327). New York: New York University Press.

van der Kolk, B., & Sapporta, J. (1993). Biological response to psychic trauma. In J. P. Wilson & B. Raphael (Eds.), *International handbook of traumatic stress syndromes* (pp. 25–35). New York: Plenum Press.

Wang, S., Wilson, J. P., & Mason, J. (1996). Stages of decompensation in combat related PTSD: A new conceptual model. *Integrative Physiological and Behavioral Science, 31*(3), 237–253.

White, R. W. (1959). *The ego and reality in psychoanalytic theory.* New York: International Universities Press.

Williams, M. B., & Sommer, J. F., Jr. (Eds.). (1994). *Handbook of posttraumatic therapy.* Westport, CT: Greenwood Press/Praeger.

Wilson, J. P. (1980). Conflict, stress and growth. In C. R. Figley & S. Leventman (Eds.), *Strangers at home* (pp. 123–165). New York: Praeger.

Wilson, J. P. (1981). *Cognitive control mechanism in stress response syndromes and their relation to different forms of the disorder.* Paper presented at the Hospital and Community Psychiatry Conference, San Diego, CA.

Wilson, J. P. (1988). Understanding the Vietnam veteran. In F. Ochberg (Ed.), *Posttraumatic therapy and victims of violence* (pp. 227–254). New York: Brunner/Mazel.

Wilson, J. P. (1989). *Trauma, transformation and healing: An integrative approach to theory, research and post-traumatic therapy.* New York: Brunner/Mazel.

Wilson, J. P. (1994, October). Historical evolution of the diagnostic criteria for posttraumatic stress disorder—From Freud to DSM-IV. *Journal of Traumatic Stress, 7*(4), 681–698.

Wilson, J. P. (1995). Traumatic events and PTSD prevention. In B. Raphael & G. Barrows (Eds.), *The handbook of preventative psychiatry* (pp. 281–296). Amsterdam: Elsevier.

Wilson, J. P., Harel, Z., & Kahana, B. (Eds.). (1988). *Human adaptation to extreme stress: From the Holocaust to Vietnam.* New York: Plenum Press.

Wilson, J. P., & Lindy, J. D. (Eds.). (1994). *Countertransference in the treatment of PTSD.* New York: Guilford Press.

Wilson, J. P., & Moran, T. (1997). Psychological trauma: PTSD and spirituality. *Journal of Psychology and Theology, 26*(2), 168–178.

Wilson, J. P., & Raphael, B. (Eds.). (1993). *International handbook of traumatic stress syndromes.* New York: Plenum Press.

Wilson, J. P., & Thomas, R. (1999). *Empathic strain and countertransference in the treatment of PTSD.* Paper presented at the 14th annual meeting of the International Society for Traumatic Stress Studies, Miami, FL.

Wilson, J. P., & Zigelbaum, S. D. (1986). PTSD and the disposition to criminal behavior. In C. R. Figley (Ed.), *Trauma and its wake* (Vol. II, pp. 305–321). New York: Brunner/Mazel.

Yehuda, R. (Ed.). (1998). *Psychological trauma.* Washington, DC: American Psychiatric Press.